



# Diagnosis and Management of Hypothermia in Neurosurgical Patients

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Dear Colleagues:

I came across an article that I wanted to share with you that was put together by Powers and Friedman out of Duke University Department of Neurosurgery regarding hyponatremia. It is in Contemporary Neurosurgery. I found it to be extremely helpful in the management of hyponatremia.

Hyponatremia is a common complication of subarachnoid hemorrhage, brain tumors and cerebral infections. Hormonal factor such as Natriuretic Peptides and Antidiuretic Hormone play important roles in hyponatremia and neurosurgical patients. It is important to differentiate between cerebral salt-wasting (CSW) and syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

## Epidemiology:

Hyponatremia is estimated to occur in 6-22% of all hospitalized patients, making it the most common electrolyte abnormality in the hospital setting. In the intensive care unit it occurs in as many as 30% of patients. As many as 35% of patients with subarachnoid hemorrhage suffer from hyponatremia. Powers and Friedman also mention from their research that approximately 23 to 25% of patients develop late hyponatremia following transsphenoidal pituitary surgery. Meningitis is associated with an incidence of hyponatremia of up to 70%.

Hyponatremia can present with headache, nausea, vomiting, confusion and lethargy. Patients may become obtunded and develop respiratory insufficiency, bradycardia, and hemodynamic instability. Untreated severe hyponatremia may result in seizures, cerebral edema, coma and death. It's also been identified as a cause of refractory increased intracranial hypertension following traumatic brain injury. Cerebral infarction following subarachnoid hemorrhage occurs 3 times as often in hyponatremic patients as in patients with normal sodium levels. The hospital mortality of patients with hyponatremia has been shown to be as much as 60 times higher than that of patients with normal serum sodium levels.

## Basic pathophysiology:

Hyponatremia has been defined as a serum sodium level of less than 130 to 135 milliequivalents/liter. Mild hyponatremia is a serum sodium less than 135 milliequivalents/liter and severe hyponatremia is a serum sodium less than 125 milliequivalents/liter. The most common causes of hyponatremia in a neurosurgical patient is cerebral salt-wasting and SIADH. The most reliable way to differentiate these causes is to determine the volume status. Patients with cerebral salt-wasting are often volume depleted whereas patients with SIADH are normal volumic. There is a theory that states that the brain can adapt to changes in serum sodium given enough time.

This theory has been reinforced by observation that in many cases the rate of change of serum sodium concentration is more important than the actual magnitude of change and is more strongly equated with morbidity and mortality. We know that



loss of intracellular solutes also play a major role in this process. A very nice schematic was presented by Powers and Friedman that I would like to reiterate here.

Again, the serum sodium level being less than 135 milliequivalents/liter for satisfying the diagnosis of hyponatremia.

First, we look at the plasma osmolality:

1. If the plasma osmolality level is 280 to 295 milliosmole/kg, then there is isotonic hyponatremia and this may be due to paraproteinemia or hypertriglyceridemia.
2. If the plasma osmolality is greater than 295 milliosmole/kg then there is hypertonic hyponatremia. This may be due to hyperglycemia or Mannitol therapy.
3. If the plasma osmolality is less than 280 milliosmole/kg then one may have hypotonic hyponatremia.

Next, the urine osmolality is measured:

1. If the urine osmolality is less than 100 milliosmole/kg then one has excessive water intake and this is due to psychogenic polydipsia.
2. If the urine osmolality is greater than 100 milliosmole/kg then there is impaired renal concentration (SIADH, CSW, etcetera).

Next, the volume status has to be assessed:

1. If the volume status is normal, then there is SIADH, an endocrinopathy, or potassium loss.
2. If the volume status is decreased, check the urine sodium.
3. If the urine sodium demonstrates greater than 20 milliequivalents/liter then there is renal solute loss which may be due to cerebral salt-wasting, diuretics or Addison's disease.
4. If the urine sodium is less than 10 milliequivalents/liter then there is extrarenal solute loss. This could be due to gastrointestinal tract loss or skin losses.
5. If the volume status is increased and the urinary sodium is greater than 20 milliequivalents/liter, then there is renal failure.
6. Urinary sodium that is less than 10 milliequivalents/liter is consistent with edematous states, such as CHF and cirrhosis.

One of the key factors in determining the causes and management of hyponatremia in patients is urine sodium status.

## Treatment:

As you can see, one treatment of hyponatremia in the neurosurgical patient is based on diagnosing its cause. For instance hyponatremia secondary to steroid hormone deficiency or hypothyroidism is managed by appropriate hormonal replacement. Fluid restriction is the first line therapy for SIADH. Aggressive fluid resuscitation is important for cerebral salt-wasting. Hypertonic saline is used for patients with cerebral salt-wasting (the sodium must be checked frequently). If there is too rapid a correction of the sodium, then one can develop central pontine myelinolysis. Powers and Friedman recommend that oral administration of sodium chloride may be an appropriate alternative in conscious patients who don't have severe cerebral salt-wasting.



Also, exogenous administration of mineral corticoid, fludrocortisone acetate, is becoming a method of preventing cerebral salt-wasting in patients with subarachnoid hemorrhage. They quote a randomized controlled trial of patients with aneurysmal subarachnoid hemorrhage and found that hyponatremia occurred in only 6% of patients receiving fludrocortisone acetate compared with 33% without fludrocortisone acetate. They also mention a potential role for direct ADH receptor antagonists. They quote several studies looking at an oral V2 receptor antagonist which is called Lixivaptan (VPA-985). This receptor antagonist causes a dose-dependent decrease in urinary sodium excretion with a concomitant improvement in hyponatremia in patients with SIADH.

In summary, fluid status, urinary sodium levels, and volume status are key for the effective management of hyponatremia. Powers and Friedman recommend that after the cause of hyponatremia has been established, then chronic hyponatremia is corrected gradually over several days. Acute hyponatremia is corrected more rapidly but should not exceed a rate of 12 milliequivalents/liter over 24 hours. Patients with mild hyponatremia due to SIADH are treated with fluid restriction of less than 1 liter per day. Their serum sodium should be monitored every 12 hours. Patients with severe hyponatremia due to SIADH are fluid restricted and can also be given sodium chloride in the form of oral salt tablets (1-2 grams with meals) or hypertonic saline (1.8% at 50 ml/hr). Patients with mild hyponatremia due to cerebral salt-wasting or extrarenal sodium loss are volume resuscitated with normal saline typically at a rate of 100 to 125 ml/hr. Patients with severe hyponatremia due to salt-wasting or extrarenal sodium loss are treated with infusion of hypertonic saline (1.8% at 50 to 100 ml/hr). Sodium chloride oral tablets may also be utilized as well. It's important to monitor closely the results of this therapy with serum sodium levels checked every 6 hours to avoid too rapid correction of hyponatremia.

In conclusion, hyponatremia complicates and is a complication of many surgical problems. This knowledge of the appropriate management of hyponatremia allows the clinician to make appropriate decisions on effective treatment strategies and avoid useless therapies and potentially lethal complications.

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